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Posttraumatic Stress Disorder and Metabolic Syndrome: Retrospective Study of Repatriated Prisoners of War

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ABSTRACT Objective: We conducted a retrospective study of metabolic data for Vietnam-era repatriated prisoners of war (RPWs) and a comparison group to determine if metabolic syndrome (MbS) was more common in those individuals with clinically diagnosed, current or lifetime posttraumatic stress disorder (PTSD) as suggested in a recent report. Methods: The metabolic data of our patients nearest the time of psychiatric evaluation (1998–2004) for PTSD were analyzed using both an analysis of variance and logistic regression. Results: Although we found elevated triglyceride levels (40 mg/dl higher) in RPWs with PTSD who met MbS criteria, overall the prevalence of MbS was the same in RPWs with and without PTSD and comparison group. Moreover, current PTSD symptom severity did not increase the likelihood of MbS. Conclusions: Our results from these repatriates who actively participate in a 37-year medical follow up program do not support the conclusion that MbS occurs more commonly in individuals with current PTSD.

INTRODUCTION

Metabolic syndrome (MbS) also known as insulin resistance syndrome and syndrome X was first described by Reaven in 1988.[1,2] There are at least 6 different definitions of MbS but all consist of a combination of obesity (increased waist circumference, waist-to-hip ratio, or body mass index [BMI]), hypertension, insulin resistance, low high density lipoprotein (HDL) cholesterol (HDL-C), elevated triglycerides (TGs), and impaired fasting glucose or impaired glucose tolerance.[3] MbS is a common syndrome, and it occurs in 22% of the young-adult (20–29 years old) population overall with an age-dependent increase to 43% in senior-aged adults (60–69 years old), as demonstrated in the third National Health and Nutrition Examination Survey.[4]

This constellation of metabolic disorders described above increases the likelihood of future type 2 diabetes by 9- to 34-fold,[5] and several meta-analyses have shown that MbS increases the future incidence of cardiovascular events.[6–8] Other studies, however, have reported a weak association between MbS and cardiovascular morbidity.[9] In the absence of diabetes, the 10-year risk for coronary heart disease events is only mildly elevated.[10] Furthermore, several authorities argue that the MbS does not confer risk of cardiovascular events beyond its individual components.[11,12]

Abdominal obesity and insulin resistance are major risk factors for the development of MbS, and genetic studies show a strong tendency to inherit MbS in offspring.[13] Additional risk factors that may have an influence are socioeconomic status and educational level, which have been shown to be inversely correlated with MbS.[14,15] Research suggests that psycho-social stress plays a role in the development of the MbS.[16–18] Several studies have demonstrated an association between combat-related posttraumatic stress disorder (PTSD) and MbS. Jakovljević et al[19] demonstrated an increased incidence (31.9%) of MbS in Croatian war veterans with PTSD, compared to 8.6% reported in the general population. In 2009, Heppner et al reported an association of PTSD and MbS in a cohort of middle-aged primarily male American veterans. This study group had a high prevalence of PTSD (55%), major depression (64%), and MbS (40%).[20] Depression was not a significant predictor of MbS risk in this cross-sectional study, but it was in a second study by Jakovljević et al.[21]

Several studies have also shown an association between combat-related PTSD and cardiovascular disease, but they did not evaluate a possible association with MbS.[22,23] The purpose of this study was to test the following hypothesis: the prevalence of MbS will be higher in repatriated prisoners of war (RPWs) with PTSD than in RPWs without PTSD or a matched comparison group (CG) without PTSD who had experienced combat but were never prisoners of war. Like the Heppner et al. study, we also hypothesize whether greater severity of current PTSD will be associated with a higher likelihood of MbS.

METHODS

A cross-sectional analysis of the metabolic data collected nearest the time of psychological evaluation was conducted with 351 male patrons (83% former combat-experienced aviators and 17% former combat-experienced ground troops) who visit the Robert E. Mitchell Center for Prisoner of War Studies located in Pensacola, Florida. This voluntary medical and psychological evaluation program has been available to Vietnam War-era RPWs since 1973. It has also been available for those RPWs from Operation Desert Storm (1991) and Operation Iraqi Freedom (2003), but their data are not included in this report. A CG of combat experienced male aviators who were not held prisoner by the Vietnamese but were otherwise

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14. ABSTRACT

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15. SUBJECT TERMS

PTSD, prisoners of war, metabolic syndrome, diabetes

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similar to the Vietnam-era repatriates (eg, age, education, aircraft flown, combat flight hours, marital status, and rank) have also been patrons of the Center since 1976. All patrons (ie, 286 RPWs and 65 CG) in this program have an opportunity to receive a comprehensive annual physical and psychiatric evaluation. The data used for the cross-sectional analysis reported in this study is from the 1998 to 2004 time-frame, which is approximately 25 years after repatriation for the RPWs and 22 years after study enrollment for the CG. It took nearly 6 years for all active patrons to cycle through and be psychiatrically evaluated. The ages of the patrons at the time were on average 61 years (±6 years SD). All subjects signed a consent form to use their data in this study, and this research was approved by the institutional review board of the Naval Medical Center in Portsmouth, Virginia.

Medical Diagnosis

A clustering of metabolic risk factors was evaluated in the database. Specifically, data examined consisted of BMI, systolic blood (SYSBP), and diastolic blood (DIABP) pressure, HDL-C levels, TGs levels, and fasting plasma glucose concentration (ie, fasting blood sugar [FBS]). In these data, 13% of the database was missing, particularly of blood pressure data (7% missing). Of this 13%, 11% were imputed using median scores for the particular missing metabolic data on the basis of a patron's set of visits to the Center. The database also included International Classification for Diseases, 9th revision (ICD-9) diagnostic coding for hypertension as a metabolic risk marker.[24] A patron was considered at risk for MbS using a modified National Cholesterol Education Program (NCEP) definition for the Adult Treatment Panel III criteria if at least 3 of the following risk factors were met: (1) BMI of 30 kg/m^2 or higher, (2) SYSBP of 130 mm Hg or higher or DIABP of 85 mm Hg or higher, or identified through ICD-9 coding 401.xx to 405.xx as hypertensive, (3) HDL-C of 40 mg/dl or less, (4) fasting TGs of 150 mg/dl or higher, and (5) FBS of 100 mg/dl or higher. We used the newer cut-point of a fasting glucose of 100 mg/dl as recommended in the 2005 Executive Summary from the American Heart Association—National Heart, Lung, and Blood Institute.[10] Our modified NCEP definition substituted BMI for waist measurements, like in many other studies,[8] and we allowed for the ICD-9 code for hypertension to signify the existence of elevated blood pressure.

A patron's metabolic data was then "dummy" coded as "1," meeting the MbS criteria, or "0," not meeting the criteria. The dichotomous codes were later used as the outcome variable for statistical analysis purposes.

In this cross-sectional analysis (1998–2004), we excluded anyone who had been medically diagnosed with a clinical endpoint in this analysis timeframe, such as diabetes or any cardiovascular or cerebrovascular diseases, as MbS is considered a precursor to these endpoints. We also excluded all patients with comorbid psychiatric diagnoses to focus on the possible unique risk associated with PTSD. These exclusions reduced the sample for this cross-sectional study to 351 patrons. Of these 351 patrons, 107 were identified as hypertensive by ICD-9 coding and were on antihypertensive medication.

In recognition of the various selection biases[25] that are most relevant to case–control studies similar to this study, we insured that the RPWs with PTSD, RPWs without PTSD, and CG groups were as similar as possible with respect to the variables that are generally viewed as predictors of MbS. In addition, we insured that all 3 groups underwent identical clinical evaluations by the same staffs who were unaware of the purpose of this study at the time the data were collected, and all procedures used to define "caseness" and MbS diagnosis were equivalent across groups. Finally, the existence of 2 control groups (RPWs without PTSD and CG, to control for former prisoner of war status and military experience, respectively) also provided some degree of design control over systematic errors that might jeopardize the validity of this study.

Psychiatric Diagnosis

The psychiatric diagnosis was used to classify the patrons of the Robert E. Mitchell Center into 3 groups. Because these veterans had all experienced combat, the repatriates and the CG were, therefore, psychiatrically assessed in the 1998–2004 time-frame for late onset of PTSD using the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) criteria.[26] RPWs without PTSD were identified in this study as the RPW– group. Those with PTSD were the RPW+ group. Four individuals with PTSD diagnosis were excluded in the CG, as their PTSD was combat related and not from capture and imprisonment. All were also tested in the 1998–2004 time-frame with the Impact of Event Scale-Revised (IES-R; a 22-item self-report measure that assesses current subjective distress caused by traumatic events),[27] and the data was included in the statistical analysis. The IES-R provided frequency and severity data for 3 subscales (intrusions, avoidance, and arousal) and for total scale. The IES-R identified 61 RPW+ patrons who were currently symptomatic for PTSD at the time the IES-R was administered, whereas an additional 29 repatriates had lifetime PTSD diagnoses but did not meet DSM-IV criteria for current PTSD. Fortunately, the incidence of comorbid psychiatric illness was very low (n = 16) in this unique cohort, and consequently, we were able to isolate the effects of PTSD on MbS by excluding those subjects with comorbidities (resulting in a total n = 351).

Power Analysis

Statistical power analyses were conducted to determine the sample size needed from the database at a power of 0.80, which would result in a small-to-moderate effect size for each analysis. The power analysis for the analysis of variance (ANOVA) indicated that a sample size of approximately 64 patrons per group was needed to detect small effect differences (Cohen's d = 0.20) of BMI, TGs, HDL-C, and FBS. The power analysis for the logistic regression used "PASS" (Power Analysis and Sample Size for Windows 2000) and is based on Hsieh, Bloch, and Larsen[28] algorithms to obtain a predictive odds ratio at

2.5 (moderate effect size). The sample size needed to determine if MbS is more common in those with PTSD (used as a categorical variable in our logistic regression) is 158. Our study included a total of 351 patrons, a sample size well above the power estimates.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS version 17.0) software was used to conduct a series of analyses of the metabolic data (ie, BMI, FBS, TGs, HDL-C, SYSBP, and DIABP) across the 3 groups. An ANOVA was conducted followed by a series of Bonferroni post hoc tests to identify group differences in the metabolic data. A logistic regression was conducted to examine the relationship between PTSD and MbS. MbS was regressed on the following variables in 3 blocked entries to build and test each block's contribution to the model[29]: Block (1), age at time of evaluation; Block (2), education as a categorical variable into 3 groups [(a) high school, (b) college, and (3) graduate school]; and Block (3), PTSD [(a) DSM diagnosis, (b) IES-R total score indicating current PTSD symptom severity, and (c) IES-R by PTSD diagnosis interaction indicating current symptomatology with PTSD]. Before conducting the logistic regression analysis, we determined that the following assumptions had been met: independence of errors, absence of multicollinearity, and linearity of the logit. Independence of errors was affirmed by the lack of serial patient assessment. Before assessing for multicollinearity, the IES-R total score data were standardized (ie, z-scored) as a form of centering these data to avoid multicollinearity impacting the results.[30] The absence of multicollinearity was confirmed through both basic collinearity statistics (Tolerance = 0.994 and Variance inflation factor = 1.006) and additional collineraity diagnostics (near equivalence of eigenvalues and condition indices and an even distribution of variance proportions). A subsequent analysis of residuals documented the absence of outliers. Finally, the dichotomous codes for MbS were cross-tabulated (by group) and odds ratios were calculated to identify if MbS was more statistically prevalent with those with PTSD.

RESULTS

Metabolic Data Profile

Table I profiles the metabolic data across the RPW+, RPW−, and CG groups. The ANOVA revealed no significant difference among the 3 groups in BMI, HDL-C, and FBS values (Table I). However, the ANOVA did reveal statistically significant differences in TGs among the 3 groups. Bonferroni post hoc tests further revealed the RPW+ group (Mean = 175 mg/dl and SD = ±175.5) was significantly different ($p < 0.05$) in TGs to both the RPW− (Mean = 140.3 mg/dl and SD = ±77.9) and CG groups (Mean = 129.2 mg/dl and SD = ±74.9).

Table II shows the results of the development of the logistic regression predictor model for MbS. Blocks 1 (Age) and 3 (PTSD) did not adequately fit the model. Block 2 for educational categories did fit the predictive model (-2 log likelihood = 388.501, $\chi^2 = 8.981$, $p = 0.03$, Negelkerke R Square = 0.037). In regards to the education categories (Table III), college (Ed_Cat(1)) and graduate school education (Ed_Cat(2)) in comparison to high school education were significant

TABLE I. Means and Standard Deviations (SD) Across Groups and Subgroups With Statistical Results of the Metabolic Data at the Group Level

Metabolic Risk Factors	Group	All			MbS					
					Present Subgroups			Absent Subgroup		
		N	Mean	SD	*N*	Mean	SD	*N*	Mean	SD
BMI	RPW+	89	27.6	3.6	26	30.4	3.7	63	26.5	2.8
	RPW−	196	27.1	3.7	47	29.8	5	149	26.2	2.6
	CG	65	26.8	3.4	15	30	2.1	50	25.8	3.1
TGs[a]	RPW+	89	175.2	175.5	27	315.3	260.3	62	114.1	55.3
	RPW−	196	140.3	77.9	47	218.1	96	149	115.7	51
	CG	65	129.2	74.9	15	211.3	94.4	50	104.5	45.9
HDL-C	RPW+	89	51.4	13.2	27	40.5	8.4	62	56.2	12
	RPW−	196	51.4	14.1	47	39.6	10	149	55.1	13.2
	CG	65	50.2	11.3	15	40.8	8.5	50	53.1	10.5
FBS	RPW+	90	97	15	27	107.3	20	63	92.6	9.5
	RPW−	196	94.2	11.1	47	102.9	13.9	149	91.5	8.4
	CG	65	95	11.1	15	100.6	10.1	50	93.3	10.9
SYSBP	RPW+	86	129.7	16	26	138.7	16.2	60	125.8	14.4
	RPW−	192	129.5	16.4	45	133.1	15.8	147	128.5	16.5
	CG	64	125.8	14.6	15	129.1	11.9	49	124.8	15.4
DIABP	RPW+	86	81.5	11.1	26	86.9	13.2	60	79.2	9.2
	RPW−	192	81.3	9.2	45	82.7	8.2	147	80.8	9.5
	CG	64	78.9	10.1	15	84.8	7.7	49	77.1	10.2

[a]ANOVA $F(2, 347) = 4.09$, $p < 0.025$; Bonferroni post-hoc RPW+ > RPW− (mean difference = 34.9, SE = ±14.2, $p < 0.05$); RPW+ > CG (mean difference = 46.0, SE = ±18.1, $p < 0.05$.

predictors for not having MbS (Ed_Cat(1) B = –0.884, Wald = 6.052, $p = 0.014$, odds ratio [Exp (B)] = 0.413 and Ed_Cat(2) B = –0.676, Wald = 3.948, $p = 0.047$, odds ratio = 0.509). Using the inverse of the odds ratios (1/0.413 = 2.42 and 1/0.509 = 1.96) suggests that those with high school education were approximately 2 times more likely to have MbS than those with higher-level education. Neither age, nor lifetime PTSD (ie, ptsd_dx) diagnosis, nor current PTSD severity (ie, Ziesr_t), nor current symptomatology with PTSD (ie, Ziesr_t by ptsd_dx) was significant predictors in the model.

Prevalence of MbS Analyses

At the time of psychiatric evaluation, 25% of the patrons of the Robert E. Mitchell Center met the MbS criterion (Table IV). The prevalence of MbS in each group underwent an odds ratio analysis, and there were no significant differences (Table V).

DISCUSSION

Other than significantly higher TG levels, RPWs diagnosed with PTSD showed no other significant metabolic differences compared to the RPWs without PTSD or the matched CG. Moreover, the data failed to show an increased prevalence of MbS in RPWs with PTSD. Our results showed that neither age nor PTSD (lifetime or current) were good fits for the MbS logistical regression model. Only education level was a significant predictor, with higher education level serving as a protective variable against the development of MbS. Educational level has been reported as a factor in other MbS research.[31,32] These findings are contrary to the Heppner et al. hypothesis of greater PTSD severity associated with greater likelihood of MbS.

TABLE II. Logistic Regression Predictive Model Development for MbS

Blocks	–2 log likelihood	χ^2	*p* Value	Nagelkerke R Square
Age	394.548	2.934	0.087	0.012
Education	388.501	8.981	0.03*	0.037
PTSD	386.199	11.283	0.08	0.047

*$p < 0.05$

Our results are different from those recently reported by Heppner in a number of ways (Table VI).[20] In our study, the prevalence of PTSD, depression, and tobacco use were much lower. In the case of alcohol abuse, which in our study was lower, we excluded these individuals altogether. Additionally, in our study, educational level and average age were significantly higher. Furthermore, we excluded those individuals with any psychiatric co-morbidities with PTSD. The Veterans Affairs study used a FBS cut-point of 110 mg/dl, although we used the newer cut-point of 100mg/dl as a criterion of MbS (as recommended in the 2005 modifications to the ATP III Guidelines),[10] which should mean more people were included. They used the waist-to-hip ratio and we used BMI. Other differences include the following: (1) we conducted a power analysis and determined we had over double the number needed to identify statistical differences. This is not clear in their report as their finding was marginally significant. (2) We did not include anyone who had reached a clinical endpoint (ie, diabetes or cardiovascular or cerebrovascular diseases). This is not clear in their report, which included World War II and Korean War veterans who in their 70s and 80s would likely have reached such clinical endpoints. (3) We accounted for those who were on an antihypertensive medication. It is unclear if medications were accounted for at all in their study. With all this, however, the most important difference between these 2 studies is the fact that our study included a matched CG who were also screened for late onset PTSD diagnosis and screened for severity of traumatic events, whether or not they had PTSD symptoms, because all had been exposed to combat stress. There was no difference in predicting MbS in either the repatriates (with or without PTSD) or the CG.

Major strengths of our study included the inclusion of a CG and a long-term relationship with the patients. Several study limitations should be noted, however. Many of the MbS studies reported in the literature used different diagnostic criteria for MbS. Even when researchers used NCEP ATP III criteria, they often modified the criteria for obesity and FBS, such that comparisons between different studies are difficult. Also, we were unable to report on medication used for hyperlipidemia, which may have had a negligible influence on our results. Most medications prescribed for hyperlipidemia are

TABLE III. Logistic Regression Education as Predictor Variable for MbS

		B	SE	Wald	df	Sig.	Exp(B)	95% CI for Exp(B) Lower	95% CI for Exp(B) Upper
Step 1[a]	AgeatExam	–0.027	0.021	1.583	1	0.208	0.973	0.933	1.015
	Ed_Cat			6.235	2	0.044*[b]			
	Ed_Cat(1)	–0.884	0.359	6.052	1	0.014*[b]	0.413	0.204	0.836
	Ed_Cat(2)	–0.676	0.34	3.948	1	0.047*[b]	0.509	0.261	0.991
	Constant	1.19	1.284	0.859	1	0.354	3.288		

Ed_Cat, education categories (0, high school; 1, college; 2, graduate school); Ed_Cat(1), college compared to high school; Ed_Cat(2), graduate school compared to high school. The SPSS table lists the b coefficients, the SE of b, the Wald statistic and its significance indicating the variable's coefficient to be significant in the equation, and the odds ratio (labeled Exp(B)) as well as 95% confidence interval (CI) on the odds ratio. [a]Variable(s) entered on step1: Ed_Cat. [b]$p < 0.05$.

targeted at elevated low-density lipoprotein cholesterol, which is not a diagnostic criterion for MbS. Although it is true that the most common drug family prescribed for hyperlipidemia (statins) can raise HDL-C, the effect is modest, 5 to 20%.[33] To evaluate this possible effect, we undertook an analysis of all current medications that might affect HDL levels. Our analysis of the use of anti-hyperlipidemic drugs (statin and niacin families) failed to show a difference between those with or without MbS.

TABLE IV. Prevalence of MbS Across the Groups

	MbS				
	Present	% Present	Absent	% Absent	Total
RPW+	27	30%	63	70%	90
RPW–	47	24%	149	76%	196
CG	15	23%	50	77%	65
Total	89	25%	262	75%	351

TABLE V. Odds Ratio Comparisons Across Groups

Comparisons	Odds Ratio	Confidence Interval		Critical z Score	*p*
		Lower Limit	Upper Limit		
RPW+ vs. RPW–	1.36	0.78	2.37	1.08	NS
RPW+ vs. CG	1.43	0.69	2.97	0.95	NS
RPW+ vs. (CG and RPW–)	1.38	0.81	2.35	1.17	NS

NS, not significant.

TABLE VI. Comparison of Two Studies

Parameter	VA Study	REMC Study
Average Age	52 (SD ± 9)	61 (SD ± 6)
Waist-to-Hip Ratio	0.97 (SD ± .07)	No data
BMI	—	27
Serum TGs (mg/dl)	190	148
HDL-C (mg/dl)	42	51
FBS (mg/dl)	106	95
Systolic Blood Pressure (mm Hg)	130	128
MbS	39.90%	25%
PTSD	55%	26%
Depression	64%	8% (excluded)
Smoking	39%	5%
Alcohol Abuse	69%	4% (excluded)
Average Educational Level (years)	13 (SD ± 2)	17 (SD ± 2)
Percentage of Military Officers	No Data	83%
Co-morbidities with PTSD	Reported	*N* = 16 (excluded)
FBS (mg/dl) Cut Point	110	100
Measure of Obesity	Waist-to-Hip Ratio	BMI
Power Analysis	Not Reported	Analysis reported (see above)
Inclusion of Participants who have Reached Clinical End Points	Not Reported	Analysis reported (see above)
Antihypertensive Medications	Not Reported	Accounted for
Inclusion of Matched CG	No	Yes

CONCLUSIONS

Despite preliminary evidence by Heppner et al., which suggests that the current PTSD symptomatology may increase the development of the MbS, our study failed to show such prevalence in RPWs with either current or lifetime PTSD compared to RPWs without PTSD and a CG. When the results of this study are combined with those of Heppner et al's study, we consider it premature to conclude that there is a clinically relevant relationship between PTSD and MbS.

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